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# Treatment of Complicated Prosthetic Aortic Valve Endocarditis With Annular Abscess Formation by Homograft Aortic Root Replacement

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**The outcome of 30 consecutive patients with active aortic prosthetic valve endocarditis and root abscesses treated by the technique of homograft aortic root replacement with reimplantation of the coronary arteries is detailed. The principles of this technique are the removal of all abscesses and infected areas likely to drain into the infected mediastinum, excision of infected tissues down to healthy noninfected tissue and replacement with an antibiotic-impregnated homograft aortic root.**

**All patients had evidence of progressive cardiac failure and ongoing sepsis. Mean patient age (± SD) at the time of operation was 42 ± 18 years. The mean number of previous aortic valve replacements per patient was 1.6 ± 0.7; 14 patients (47%) had undergone ≥2 previous replacements. At operation, aortic root abscesses were found in all patients; abscess extension to adjacent structures and partial valve dehiscence had occurred in 23. In-hospital death occurred in 9 (30%) of the 30 patients.**

**The 21 hospital survivors have been followed up for a mean of 66 ± 42 months (range 9 to 144). Overall, 17 (81%) of the 21 hospital survivors have remained free of major adverse events (recurrence of endocarditis, need for reoperation or death). The results of our study suggest that homograft aortic root replacement should be considered favorably in the treatment of patients with aortic prosthetic valve endocarditis and root abscesses.**

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Prosthetic valve endocarditis is a devastating although relatively uncommon complication of cardiac valve replacement. It complicates approximately 2% to 4% of valve replacement operations (1–5) and is associated with a mortality rate >50% (1–4). Increasing surgical expertise and the high mortality rate with medical management have led to a widespread recommendation for early valve replacement in many patients with prosthetic valve endocarditis (1,2,4,6). At times, the infective process is so destructive that simple debridement and valve replacement are not sufficient (2). This is the case in patients with destructive aortic prosthetic valve endocarditis and aortic root abscesses. A number of techniques to treat such patients have been described (7–13). In this report, we describe our experience with regard to one of these techniques, homograft aortic root replacement, in 30 consecutive patients with complicated aortic prosthetic valve endocarditis.

## Methods

**Study patients (Table 1).** Between November 1972 and August 1989, 30 patients suffering from active complicated prosthetic aortic valve endocarditis and requiring emergency surgery were selected for homograft aortic root replacement. For the purposes of this study, clinical and operative details were obtained retrospectively through review of the hospital charts.

The mean patient age (± SD) was 42 ± 18 years (range 7 to 74). Twenty patients were male, 10 female. All patients had evidence of hemodynamic decompensation with ongoing sepsis. Fever was present in 30 (100%) and newly developed aortic regurgitation in 21 (70%). Peripheral manifestations of endocarditis (defined as the presence of one or more of the following: petechiae, splinter hemorrhages, Roth spots, Janeway lesions, Osler nodes, unexplained embolus or unexplained anemia) were present in 26 (87%), splenomegaly in 16 (53%) and new high grade cardiac conduction abnormalities in 3 (10%). The mean number of previous aortic valve replacements per patient was 1.6; 14 patients (47%) had had ≥2 previous aortic valve replacements.

*The onset of prosthetic valve endocarditis was taken as the earliest of*: 1) the date of the first physician visit for an illness eventually proved to be prosthetic valve endocarditis; 2) the date of the first positive blood culture for an organism eventually shown to be the causative pathogen of prosthetic valve endocarditis; or 3) the date of hospital admission. Prosthetic valve endocarditis was defined as early when it appeared within 60 days of valve insertion and late when it occurred after 60 days.

**Operative technique.** A median sternotomy was used and the right atrial appendage and ascending aorta were cannulated for cardiopulmonary bypass. On bypass, the patient's body temperature was cooled to 28°C, the aorta was cross

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**Table 1.** Clinical Characteristics and In-hospital Outcome of 30 Patients Undergoing Homograft Aortic Root Replacement

| Pt No. | Age (yr)/ Gender | Fever | AR | PM | SM | New Cond Abnl | No. Prev Ao V Repl | Infected Aortic Prosthesis | Onset E/L | Organism Grown | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 39/M | + | + | – | – | – | 1 | Hall-Medtronic | E | *Strep viridans* | Survival |
| 2 | 73/M | + | + | + | – | – | 1 | Carpentier-Edwards xenograft | L | *Strep viridans* | Survival |
| 3 | 54/M | + | – | + | + | – | 1 | Starr-Edwards | L | *Strep viridans* | Survival |
| 4 | 26/M | + | + | + | – | – | 1 | Autograft | L | *Staph epidermidis* | Survival |
| 5 | 35/M | + | + | + | + | – | 3 | Bjork-Shiley | E | — | Survival |
| 6 | 70/M | + | – | + | – | – | 1 | St. Jude | L | *Strep fecalis* | Survival |
| 7 | 48/M | + | – | + | – | – | 1 | Carpenter-Edwards xenograft | L | *Strep viridans* | Survival |
| 8 | 45/F | + | + | – | + | – | 2 | Starr-Edwards | L | *Strep mutans* | Survival |
| 9 | 7/F | + | – | + | – | – | 1 | Starr-Edwards | E | *Staph epidermidis* | Survival |
| 10 | 40/M | + | – | + | – | – | 2 | Starr-Edwards | L | *Staph epidermidis* | Survival |
| 11 | 22/F | + | – | + | – | – | 1 | Homograft | L | *Strep sanguis* | Survival |
| 12 | 48/F | + | – | + | – | – | 3 | Bjork-Shiley | L | — | Survival |
| 13 | 30/M | + | + | + | – | – | 1 | Starr-Edwards | L | — | Survival |
| 14 | 38/F | + | + | + | + | – | 2 | Starr-Edwards | L | *Staph epidermidis* | Survival |
| 15 | 36/M | + | + | + | + | – | 1 | Starr-Edwards | L | *Staph aureus* | Survival |
| 16 | 40/M | + | + | + | + | – | 2 | Starr-Edwards | L | *Staph aureus* | Survival |
| 17 | 41/F | + | + | + | + | – | 1 | Homograft | L | *Staph epidermidis* | Survival |
| 18 | 15/F | + | + | + | + | – | 1 | Carpentier-Edwards xenograft | L | *H parainfluenza* | Survival |
| 19 | 59/M | + | + | + | + | – | 2 | Carpentier-Edwards xenograft | L | *Strep mitior* | Survival |
| 20 | 51/M | + | + | + | + | – | 2 | Starr-Edwards | L | *Strep viridans* | Survival |
| 21 | 63/F | + | + | + | + | – | 2 | Carpentier-Edwards xenograft | L | *Strep mutans* | Survival |
| 22 | 35/M | + | + | – | + | – | 1 | Hall-Kaster | E | *Staph epidermidis* | Death |
| 23 | 55/M | + | – | + | + | – | 2 | Starr-Edwards | E | *Staph epidermidis* | Death |
| 24 | 46/M | + | – | + | – | – | 2 | Pericardial xenograft | E | *Staph epidermidis* | Death |
| 25 | 14/F | + | + | + | – | CHB | 3 | Bjork-Shiley | L | *Strep mitis* | Death |
| 26 | 28/M | + | + | + | – | CHB | 1 | Bjork-Shiley | E | *Staph aureus* | Death |
| 27 | 59/F | + | + | – | + | CHB | 2 | Medtronics | L | *Strep viridans* | Death |
| 28 | 57/M | + | + | + | – | – | 1 | Carpentier-Edwards xenograft | E | *Staph epidermidis* | Death |
| 29 | 15/M | + | + | + | + | – | 1 | Carpentier-Edwards xenograft | E | *Staph epidermidis/ Staph aureus* | Death |
| 30 | 74/M | + | + | + | + | – | 2 | Starr-Edwards | L | *Staph epidermidis/ Staph aureus* | Death |

AR = aortic regurgitation; CHB = complete heart block; E/L = early/late onset of prosthetic valve endocarditis; F = feminine; *H* = *Haemophilus*; M = masculine; New Cond Abnl = new high grade conduction abnormalities; No. Prev Ao V Repl = number of previous aortic valve replacements; PM = peripheral manifestations of endocarditis; Pt. = patient; SM = splenomegaly; *Staph* = *Staphylococcus; Strep* = *Streptococcus*; – = absent; + = present.

clamped and an oblique aortotomy was made extending down into the sinus of the noncoronary cusp. The coronary arteries were cannulated during dissection to prevent embolization by debris. After removal of the infected prosthesis and excision of the necrotic tissue down to healthy tissue, a homograft aortic root was inserted and the coronary arteries were reimplanted into the homograft. The infected bed of the previous valve site was treated with weak iodine solution.

The upper limit of the aortic incision was ≥1 cm above any evidence of infected tissue, and the lower extent of the incision was across the outflow tract of the left ventricle down to healthy tissue and generally at the point of insertion of the right coronary cusp into the septum anteriorly and across the subvalvular curtain of the mitral valve posteriorly. In the angle between the right and noncoronary cusps, the incision was extended to the upper margin of the membraneous septum to avoid damage to the conducting systems. The coronary orifices were preserved with a 3 mm cuff of surrounding aortic wall. Once both coronary arteries had been identified, a curved probe was passed into them to identify their direction during subsequent mobilization. All abscess cavities were widely opened and allowed to drain into the surrounding mediastinum.

Multiple, interrupted 4-0 Prolene sutures were then at-

tached to the lower margin of the horizontally trimmed homograft and the corresponding areas of the healthy aortic root tissues. At least 28 sutures were required, dividing the circumference into four sections with 7 to 8 sutures each. It was convenient to tie down the sutures over a thin strip of autogenous pericardium to aid hemostasis and avoid cutting through the muscles of the homograft. The left coronary artery and its disc of aortic wall were then attached to the corresponding excised orifice in the left coronary sinus of the homograft with a continuous 6-0 Prolene suture.

The upper margin of the homograft was trimmed and sewn to the transected aorta with continuous 4-0 Prolene sutures, with a temporary clamp on the right coronary ostium of the homograft. The aortic clamp was then released and the new aortic root was observed under pressure, enabling hemostasis to be checked and indicating the best site of attachment of the right coronary artery. This was a necessary precaution because the position of this artery is variable and might not correspond with the appropriate orifice in the homograft. The aortic clamp was then reapplied and the right coronary artery was anastomosed.

**Patient follow-up.** This was achieved by annual clinical examination and by telephone or postal questionnaire. Each patient routinely had a 12 lead electrocardiogram (ECG) at rest, chest X-ray film and Doppler echocardiographic study on an annual basis.

**Statistical analysis.** Wilcoxon's rank test, Student's *t* test and Fisher's exact test were performed to determine possible differences in clinical characteristics between the patients who died in hospital and those who survived. A p value $<0.05$ was considered statistically significant.

## Results

**Infected prosthesis and microbiology (Table 1).** Preoperative isolation of the infecting organism was obtained in 23 of the 30 patients. In a further four patients, the infecting organisms were identified postoperatively either from the excised prosthesis or from vegetations. Staphylococcus was identified as the causative organism in 14 patients; in 9 of these 14 patients *S. epidermidis* alone was isolated and in 3, *S. aureus* alone. In the remaining two patients with staphylococcal infection, there was a mixed growth of *S. aureus* and *S. epidermidis*. Streptococcus was the infecting organism in 12 patients and *Haemophilus parainfluenza* in 1. Prosthetic valve endocarditis was judged as late in onset in 21 patients and early in 9 (Table 1). Infection occurred on a mechanical aortic valve prosthesis in 19 patients and on a bioprosthesis in 11.

**Operative findings.** At operation, aortic annular abscesses were found in all patients. In 23 patients, the ring abscesses burrowed into adjacent structures (extending into the periaortic space in 12 patients, the left or right atrium in 10 and the interventricular septum in 3 and burrowing through the anterior mitral valve cusp in 4). Twenty-three of the 30 aortic valve prostheses were noted to be partially detached.

**Table 2.** Comparison of Clinical Characteristics of 21 Patients Surviving and 9 Not Surviving Homograft Aortic Root Replacement

| | In-Hospital Death | Hospital Survivors | p Value |
|---|---|---|---|
| Age (yr) | 43 ± 21* | 42 ± 17* | NS |
| Male (no.) | 7 | 13 | NS |
| No. of previous aortic valve replacements | 1.67 ± 0.7* | 1.52 ± 0.68* | NS |
| Early onset | 6 | 3 | <0.02 |
| New onset aortic regurgitation | 7 | 14 | NS |
| New high grade cardiac conduction abnormalities | 3 | 0 | <0.05 |
| Mechanical prosthesis | 6 | 13 | NS |
| Staphylococcal etiology | 7/9 | 7/18† | 0.13 |

*Mean ± SD; †infecting organism identified in only 18 of the 21 hospital survivors.

**In-hospital death.** This occurred in 9 (30%) of the 30 patients. Five of these patients had been transferred to our care from other cardiac surgery centers after unsuccessful surgical-medical (four patients) or medical (one patient) treatment of endocarditis. Of these five patients, three were in extremis with advanced cardiac failure and established renal failure and two had complete heart block. Three patients died in the operating room. Of the remaining six in-hospital deaths, four occurred between 3 and 14 days postoperatively as a result of progressive heart failure (combined with progressive renal failure in two). The remaining two patients died 4 and 8 weeks after surgery; one died of uncontrolled septicemia, the other of massive bleeding from a false aneurysm originating from a proximal suture line defect.

**Predictors of unfavorable outcome (Table 2).** The clinical characteristics of the group of patients who died in the hospital and those who survived the hospital stay are compared in Table 2. There were two statistically significant predictors of in-hospital death: 1) early onset of prosthetic valve endocarditis ($p < 0.02$), and 2) recent onset high grade cardiac conduction defects ($p < 0.05$). A staphylococcal etiology was identified in 7 (78%) of the 9 patients who died in the hospital, but in only 7 (39%) of the 18 hospital survivors in whom an organism was isolated ($p$ = NS).

**Out-of-hospital course.** The 21 hospital survivors have been followed up for a mean of 66 ± 42 months (range 9 to 144). During this period, the following events occurred:

*Recurrence of endocarditis.* This occurred in 2 (9.5%) of the 21 patients. One patient (Patient 18) developed endocarditis 5 years after aortic root replacement. The organism isolated (a streptococcus) was different from the organism responsible for this patient's original prosthetic valve endocarditis. She was successfully treated with medical therapy alone. The second patient (Patient 5), who developed recurrence of endocarditis 8 months after aortic root

replacement, underwent successful reoperation (repeat homograft aortic root replacement). In this patient, no organism had been identified when the first homograft aortic root was inserted, but at the time of repeat operation, laboratory investigations diagnosed Q fever (*Coxiella burnetti*) endocarditis.

*Need for repeat operation.* This occurred in four patients. One patient (Patient 5) underwent repeat aortic root replacement for recurrence of endocarditis. Two patients (Patients 18 and 14) who had primary tissue failure were reoperated on 7.5 and 10 years, respectively, after initial aortic root replacement. The first of these two patients was the patient, previously mentioned, whose recurrence of endocarditis was successfully treated medically. In both patients, the predominant lesions were calcification of the homograft wall and extension of the calcification to the cusps. In neither patient was aneurysmal dilation of the homograft or stenosis of the coronary ostia encountered. The fourth patient (Patient 16), who required reoperation as a result of progressive cardiac failure, underwent cardiac transplantation 9 years after aortic root replacement.

*Death.* This occurred perioperatively in the two patients undergoing reoperation for primary tissue failure. There were no other deaths, either cardiac or noncardiac.

*Patients who have remained event free.* Among the 17 patients who have remained event free, follow-up study to at least May 1990 has been achieved in all. Two of these 17 patients, both aged >70 years and with previous symptomatic valvular heart disease, have moderately severe (functional class III) symptoms of heart failure. At a mean follow-up interval of 61 ± 43 months, the other 15 are either asymptomatic or only mildly symptomatic. One of these 15 patients has developed aortic regurgitation that is asymptomatic and graded as mild to moderate by Doppler echocardiography.

## Discussion

Our study examined the daunting therapeutic challenge posed by patients with active aortic prosthetic valve endocarditis. In such patients, aortic root abscesses, often with associated valve dehiscence and abscess extension to adjacent structures, are virtually the rule (14). Indeed, all of our 30 patients had ring abscesses that extended to adjacent structures in 23. Given the uniform clinical picture of progressive cardiac failure and ongoing sepsis in these patients, urgent surgical intervention was clearly indicated in all. However, the operative findings in these patients made it equally obvious that simple replacement of the infected prosthesis would not eradicate the infection and more radical surgery was required.

**Surgical approaches used in the treatment of aortic prosthetic valve endocarditis and annular abscess formation.** Several innovative surgical techniques have been applied to small numbers of patients with aortic prosthetic valve endocarditis and left ventricular-aortic discontinuity. Symbas et al. (7) reported successful patch closure of the abscess and in situ valve replacement in one patient. Reitz et al. (8) described successful treatment of three of four patients by translocation of the aortic valve, closure of the native coronary ostia and placement of saphenous vein bypass grafts to the coronary arteries. Frantz et al. (9) used composite prosthetic valve-woven Dacron tube graft reconstruction of the aortic root to treat two patients (with success in one). This latter technique was also successfully applied by Van Hooser et al. (10) in three patients with prosthetic valve endocarditis and aortic root abscesses. Kirklin et al. (11) reported the successful treatment of a patient with infection of an aortic mechanical valve and an annular abscess using an aortic valve homograft placed below the level of the coronary ostia.

Despite the favorable results obtained with these techniques (7–11), it is often insufficient just to replace the infected prosthesis and attempt to paper over the pus and infected material with pericardial patches or Dacron material. To do so is to ignore the hallowed surgical rubric: "do not let the sun set on undrained pus." Simply covering over infected material and cavities creates a slowly developing infected focus that will continue to fester, grow in size and eventually burst into the area of least resistance (which is generally back into the bloodstream), possibly months later (15). The technique performed in our study patients, homograft aortic root replacement, avoids these problems. First performed by Ross (15) in 1972, this technique excludes the root abscesses and the weakened infected aortic anulus from high systemic pressures and permits suturing to a bed of the aortic root in continuity with healthy myocardium (12,13,15).

**Outcome in complicated aortic prosthetic valve endocarditis.** In our study, the technique of homograft aortic root replacement was used in 30 critically ill patients with aortic prosthetic valve endocarditis and root abscesses. In-hospital death occurred in 9 (30%). Of the 21 hospital survivors, at a mean follow-up interval of 66 ± 42 months, 17 (21%) have remained free of major adverse events.

Taking into account the clinical presentation of our patients, an in-hospital mortality rate of 30% is not surprising. The mean number of previous aortic valve operations per patient was 1.6 ± 0.7, with 14 (47%) having had ≥2 aortic valve operations. These features alone would have put them at a considerably increased surgical risk (16). In addition, all had hemodynamic decompensation and active endocarditis, many with multisystem failure. Of the nine patients who died perioperatively, three were in extremis on arrival in the operating room with cardiogenic and septic shock. In our study, early onset prosthetic valve endocarditis and new onset complete heart block emerged as predictors of in-hospital death. The higher mortality rate in patients with early as opposed to late prosthetic valve endocarditis observed in our series has also been found in all major studies (2) examining prognostic features in prosthetic valve endocarditis. This observation most likely reflects the more

virulent organisms associated with early as opposed to late prosthetic valve endocarditis (in our series, compared with the patients with late onset endocarditis, staphylococcus was isolated in proportionately twice as many patients with early onset endocarditis) and debilitation of patients after operation (2). New onset complete heart block suggests extensive abscess extension and infiltration and thus the poor prognosis in patients developing this rhythm disturbance is not surprising.

**Previous studies.** Reported large series regarding the fate of patients with prosthetic valve endocarditis treated surgically contain a considerable case mix. In these studies, patients with aortic, mitral, complicated and uncomplicated prosthetic valve endocarditis are not clearly differentiated. However, Masur and Johnson (4) reported the alarming observation that of 14 patients with aortic prosthetic valve endocarditis treated with a combined medical-surgical approach, only 1 survived. Ivert et al. (3) reported on 33 patients with prosthetic valve endocarditis (location not detailed) treated surgically at their center. The overall in-hospital mortality rate was 36% and 11 (44%) of 25 patients surviving the first repeat operation needed two or more repeat operations. Two of the 11 patients who underwent a second repeat operation had certain recurrent or residual prosthetic valve endocarditis and 1 had possible prosthetic valve endocarditis. Of the four patients in the series of Ivert et al. (3) undergoing a third repeat operation, one had certain prosthetic valve endocarditis and one had possible prosthetic valve endocarditis. Of 21 patients surviving one or more repeat operations and discharged from the hospital, 3 (14%) died of another recurrence of prosthetic valve endocarditis and another 2 died of heart failure.

Although the study of Ivert et al. (3) was not limited to patients with aortic prosthetic valve endocarditis, their results highlight the notorious tendency for prosthetic valve endocarditis to recur and the considerable in-hospital and late mortality rates associated with this condition. Accordingly, the relatively low rate of adverse events at intermediate- to long-term follow-up study observed in our series is encouraging. Given the very septic state of our study patients at the time of presentation, the rate of recurrence of endocarditis after homograft aortic root replacement (2 of 21 patients) is low and a gratifying feature of this radical operation. In contemplating the relatively low endocarditis recurrence rate in our series, a relevant consideration is that homograft valves, as opposed to other bioprosthetic or mechanical valves, are reported (17) to be more resistant to infection, at least during the first few years after implantation. A further important consideration is that none of our patients were known intravenous drug abusers.

**Limitations of the present study.** A potential limitation of our study is that comprehensive data regarding the long-term (≥10 years) consequences of use of homograft aortic root replacement in patients with complicated aortic prosthetic valve endocarditis cannot be provided because only four of the hospital survivors in our series underwent the procedure during or before 1980. However, the results of several reported studies (18,19) involving large series of patients undergoing this operation for conditions in addition to prosthetic valve endocarditis (such as for relief of various types of left ventricular outflow tract obstruction) suggest that the homograft aortic root is a very durable structure. This observation is not particularly surprising if one realizes that the technique leaves the aortic valve undisturbed within its native tubular structure and does not require any geometric wizardry to match it to a new home (20). This results in the invariable insertion of a competent mechanism that is undisturbed by surrounding tissue and that causes no turbulence. Indeed, the probability of freedom from valve failure in the homograft root has been estimated at about 92% at 10 years (21). Of course, in many centers there is limited availability of homograft tissue.

**Conclusions.** Homograft aortic root replacement appears particularly useful when it is difficult to place a conventional homograft or autograft because there has been ring destruction by infection. On the basis of the encouraging results observed in our patients, we suggest that this technique should be considered favorably in selected patients with active aortic prosthetic valve endocarditis and annular abscess formation.

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